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Case report

Stenting of right coronary ostial occlusion due to thrombosed type A aortic dissection: One-year follow-up results

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Summary A 52-year-old man experienced acute chest pain and was transferred to our hospital. An electrocardiogram showed ST-segment elevation in leads II, III, aVf, and V1 through V3. The diagnosis at the emergency room was inferior acute myocardial infarction (AMI), and emergent coronary angiography (CAG) was performed. While CAG showed subtotal occlusion of the right coronary artery (RCA) ostium, aortic dissection was suspected due to staining of the contrast agent distal to the occluded site of RCA. Intravascular ultrasound showed compression of the RCA ostium due to aortic dissection. We performed bare metal stent implantation, and contrast-enhanced computed tomography (CT) after stenting showed a thrombosed type A aortic dissection. The patient received medical treatment along with repeated CT and echocardiographic examinations, and was discharged without any events one month after admission. CAG six months after stenting and 64-multislice CT angiography one year later showed a patent RCA. Contrast-enhanced CT at six months showed complete resorption of the ascending aortic intramural hematoma, and 64-multislice CT at one year showed a descending aortic intramural hematoma. The patient is doing well one year after the onset. This is a rare case of successful medical treatment for acute type A aortic dissection complicated with AMI.

Introduction

Acute type A aortic dissection sometimes involves the ostium of the coronary artery, and leads to acute myocardial infarction (AMI). The frequency of AMI due to aortic dissection has been reported to be 1—3% [1,2]. It is difficult to make a differential diagnosis between AMI due to thrombotic occlu-

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sion of a coronary artery and AMI subsequent to an aortic dissection. Delay of diagnosis and unstable hemodynamics may often lead to a poor outcome. In previous case reports, successful treatment of type A aortic dissection combined with AMI has required emergent surgery, but our case had a good outcome without surgery. Therefore, we report this rare case and present the one-year follow-up results.

Case report

A 52-year-old man with a history of hypertension experienced severe chest pain without back pain and was transferred to our hospital about 20 min after the symptom onset. On arrival, the patient was in cardiogenic shock, with a systolic blood pressure of 80 mmHg and heart rate of 40 beats/min, with complete atrioventricular (AV) block. Complete AV block improved spontaneously, and an electrocardiogram (ECG) showed ST-segment elevation in leads II, III, aVf, and V1 through V3 (Fig. 1). A chest radiograph showed mild cardiomegaly with a normal mediastinum. Blood tests showed a white blood cell count of 11,610/mm^3, creatine kinase of 132 IU/L with MB fraction of 35 IU/L, and troponin-T of 0.10 ng/dL. Echocardiography revealed severe asynergy of the left ventricular inferior wall, and no evidence of aortic regurgitation or pericardial effusion. The diagnosis at the emergency room was inferior AMI, and therefore emergent coronary angiography (CAG) was performed via the right femoral artery using a 5-French catheter. CAG showed subtotal occlusion of the right coronary artery (RCA) ostium (Fig. 2A). However, aortic dissection was suspected due to staining of the contrast agent distal to the occluded site of RCA (Fig. 2B). Intravascular ultrasound (IVUS) showed ostial compression of the RCA due to aortic dissection (Fig. 2C). For the prompt improvement of the cardiogenic shock, a bare metal stent was implanted as a bridge therapy for surgical repair (Fig. 2D), and after the procedure the patient's hemodynamics were soon stabilized. After emergent percutaneous coronary intervention (PCI), contrast-enhanced computed tomography (CT) showed a thrombosed type A aortic dissection (intramural hematoma) (Fig. 3A—C). The maximum size of the dissected aorta and intramural hematoma was 46.5 and 20 mm at the ascending aorta, with the entry of the dissection occur-

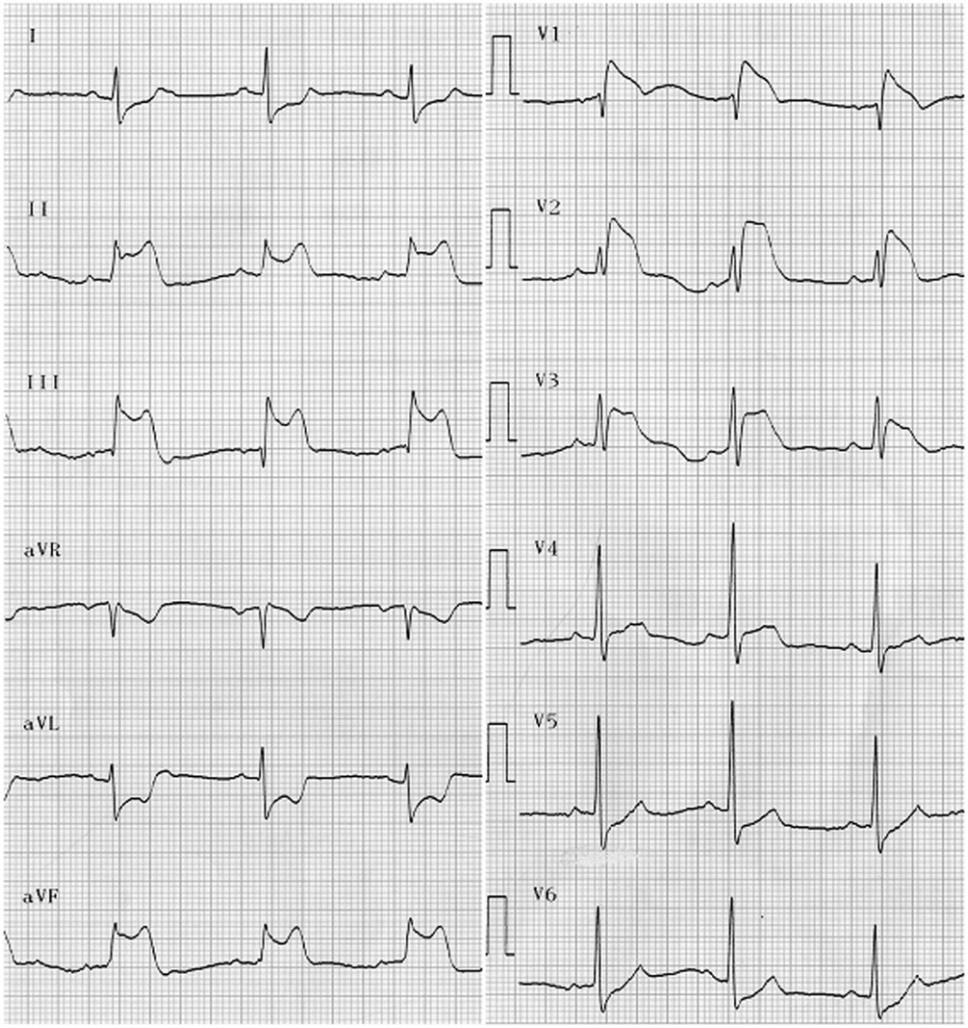

Figure 1 A 12-lead electrocardiogram showing ST-segment elevation in leads II, III, aVf, V1 through V3, and concomitant ST-depression in leads I and aVL.

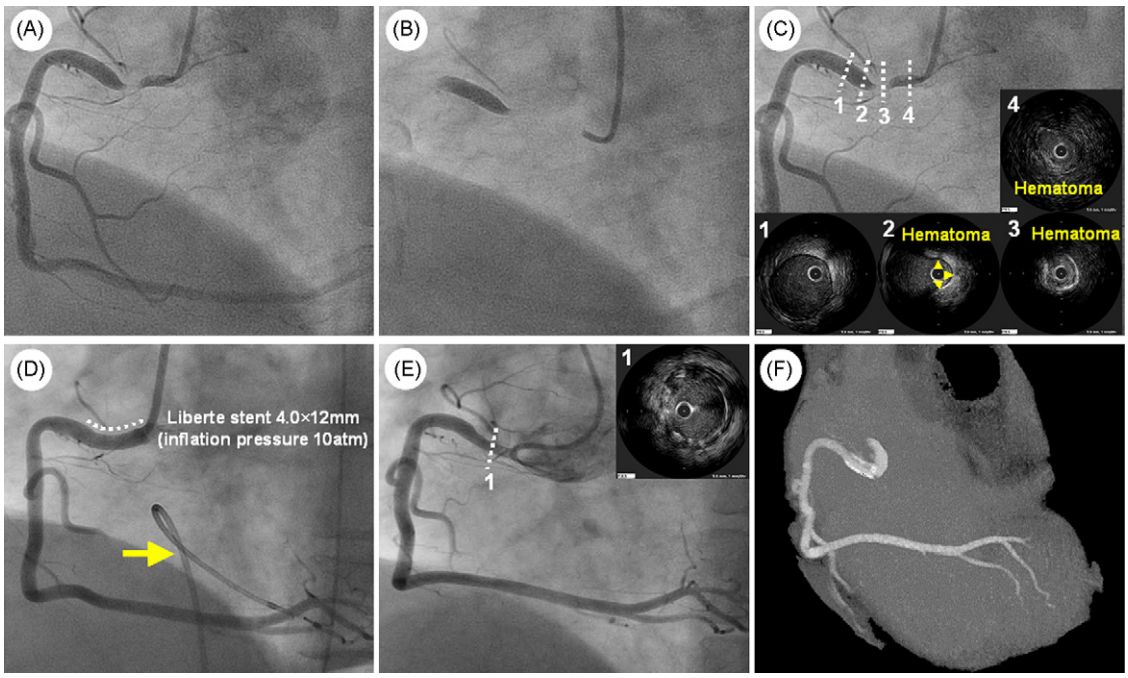

Figure 2 (A) Emergent coronary angiography (CAG) in the left anterior oblique view demonstrated subtotal occlusion of the right coronary artery (RCA) ostium. (B) Fluoroscopy post-CAG showing staining of the contrast medium. (C) Intravascular ultrasound (IVUS) images acquired at proximal locations within the RCA. (C-1) Distal reference artery beyond the stenosis showing a widely patent vessel. (C-2) The lumen was compressed to a ''potbelly'' shape by a large intramural hematoma (arrows). (C-3) Narrowest point showing collapsed vessel structure surrounded by a large hypoechoic intramural hematoma. (C-4) The RCA ostium was surrounded completely by a concentric large, homogeneous or slightly hyperechoic intramural hematoma. (D) After stenting, coronary flow was improved as assessed by thrombolysis in myocardial infarction (TIMI) 3. The arrow indicates a temporary pacing. (E) CAG at six months with no in-stent restenosis. (E-1) An IVUS image showing slight intimal proliferation, but no stent fracture or hypoechoic intramural hematoma. (F) 64-multislice CT angiography one year later with no in-stent restenosis.

ring at the distal aortic arch, as a result of the thrombosed false lumen being patent, and flow communication occurring at this point. The aortic dissection extended to the level of the bilateral common iliac arteries. However, all main branches, including the brachiocephalic artery, common carotid artery, subclavian artery, celiac artery, superior and inferior mesenteric arteries, and bilateral renal arteries, were fortunately not involved and circulated from the true lumen.

The patient received strict antihypertensive medical therapy, with continuous intravenous infusion of nitroglycerin 2 μg/kg/min for five days, atenolol (50 mg/day), controlled-release nifedipine (40 mg/day), and olmesartan (20 mg/day), in combination with antiplatelet therapy using continuous infusion of heparin (10,000 U/day for 24 h), aspirin (100 mg/day) and clopidogrel (75 mg/day). Careful imaging follow-up was performed, and progression of the intramural hematoma was not observed during hospitalization. The patient was discharged without any events one month after admission. Neither CAG and IVUS at six months after PCI (Fig. 2E) nor 64-multislice CT angiography one year later (Fig. 2F) showed in-stent restenosis. Contrast-enhanced CT at six months showed complete resorption of the ascending aortic intramural hematoma (Fig. 3D—F), and 64-multislice CT at one year showed a descending aortic intramural hematoma (Fig. 4). The patient is doing well one year after the onset.

Discussion

AMI secondary to acute ascending aortic dissection is a relatively rare, but indeed life-threatening condition. The mechanism by which dissection leads to myocardial infarction is either obstruction of the orifice of coronary arteries by a dissecting hematoma or disrupted inner layers of the aortic wall. Spittell et al. showed that dissection of the ascending aorta affects the RCA more often than the left coronary artery (LCA) [3]. Therefore, the possibility of aortic dissection should be considered in patients with an inferior AMI, particularly those with suspected right ventricular infarction or conus branch occlusion. In addition, Hirst et al. reported 39 autopsy cases of aortic dissection complicated by coronary artery dissection, and showed 18 of 39 cases involved both the RCA and LCA [4]. These data suggest that LCA is also often involved and causes cardiogenic shock or cardiac arrest due to ischemia responsible for the left main trunk occlusion. These lethal conditions sometimes mimic the true incidence of LCA involvement, and therefore when they occur, it is important to consider aortic dissection with LCA involvement and investigate this possibility using either transthoracic or transesophageal echocardiography, and chest CT prior to catheterization. In the present case, emergent CAG and enhanced CT showed no involvement of the LCA. However, an ECG on arrival showed ST-segment elevation in leads II, III, aVf, in addition to V1 through V3.

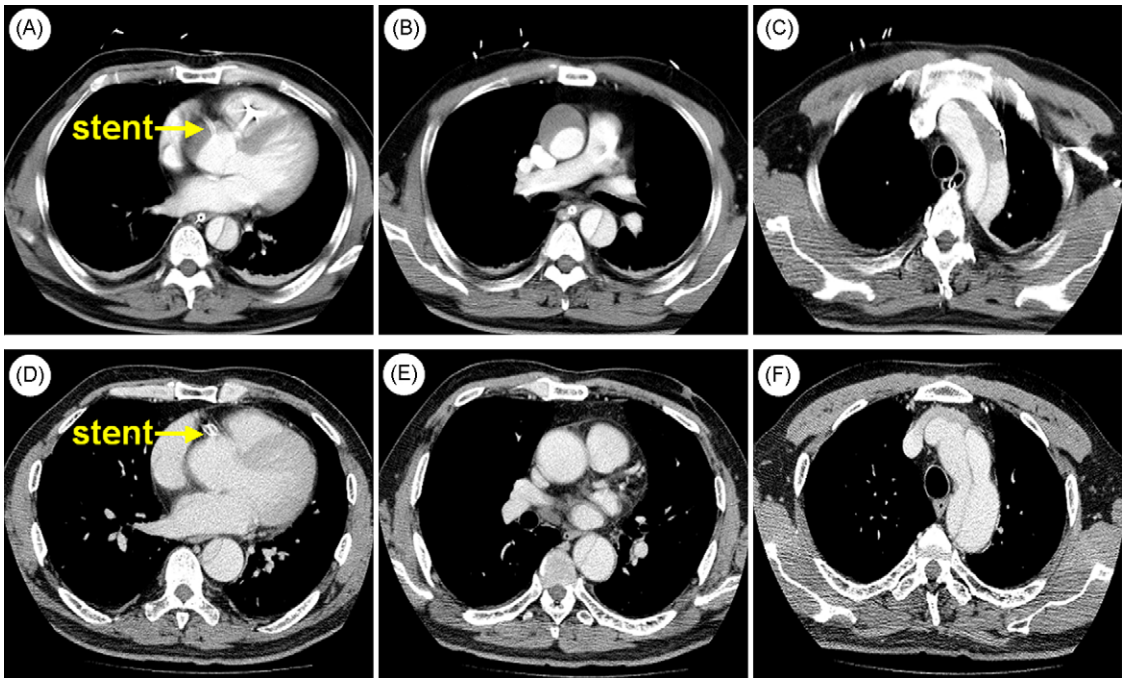

Figure 3 (A—C) Contrast-enhanced computed tomography (CT) images after stenting. (D—F) Contrast-enhanced CT images at six months. (A and D) The implanted bare metal stent (arrow) was observed clearly at the level of the right coronary cusp. (B) A chest CT scan showing characteristic crescentic wall thickening with an ascending aortic intramural hematoma and intimal flap with flow communication in the descending aorta. (C) At the level of the distal aortic arch, the thrombosed false lumen became patent with flow communication. (E) A chest CT scan showing complete resorption of the ascending aortic intramural hematoma and intimal flap with flow communication in the descending aorta. (F) A false lumen with flow communication was observed at the level of the distal aortic arch.

These findings suggest that the cardiogenic shock observed at admission may have been caused by right ventricular infarction and bradycardia, or alternatively, lethal tachyarrhythmia, as ST-segment elevation in leads V1—3 often reflects a right ventricular infarction or conus branch occlusion.

Accurate diagnosis is required for selection of appropriate and prompt treatment. The standard treatment for acute type A aortic dissection is surgery, and most clinical studies have shown a very poor prognosis of ascending aortic intramural hematoma without surgical repair [5,6]. However, recent studies have shown an excellent clinical course with mere medical treatment. Song et al. studied 41 cases of ascending aortic intramural hematoma that received medical treatment, and showed a two-year survival rate of 84%, which was not inferior to those in the surgical repair group [7]. Kaji et al. reported that survival rates in the type A aortic intramural hematoma group with supportive medical therapy were all 90% at one, two, and five years, which were significantly higher survival rates than those reported in the classic type A aortic dissection group [8].

There have been several reports about AMI caused by acute ascending aortic dissection [9,10]. However, all these cases required surgical treatment. In some cases, emergent PCI was performed as a bridge therapy before surgical repair, while surgical replacement of the ascending aorta, aortic valve resuspension, and coronary artery bypass grafting were performed in other cases. In the present case, we did not suspect aortic dissection until the emergent RCA angiography was performed. However, the CAG indicated the possibility of an aortic dissection due to staining of the contrast agent distal to the occluded site of RCA. Moreover, IVUS showed a compression of the RCA ostium due to aortic dissection. For the rapid restoration from the cardiogenic shock, we decided to perform PCI as a bridge to surgery, and planned to undertake surgery at either the acute or sub-acute phase. However, as hemodynamics stabilized after the emergent PCI, and no ischemic symptoms or complications were observed in any other organs, combined with the possibility of a worse outcome following surgery, we determined to take a strategy based on recent evidence of supportive medical therapy for acute type A aortic intramural hematoma [7,8].

To the best of our knowledge, our case is the first one of AMI caused by an acute ascending aortic intramural hematoma treated successfully by supportive medical treatment. However, there are several important issues that must be addressed in cases such as ours. The first issue is that administration of antiplatelet agents may increase the risk of aortic expansion and rupture. The second issue is how to evaluate the optimal timing of surgical treatment during the follow-up period. Our patient received an infusion of heparin and antiplatelet agents before and after the PCI. If the size of the hematoma increased or progression to overt dissection occurred, then surgical repair should have been necessary.

In conclusion, this is the first report of successful medical treatment for acute type A aortic dissection complicated

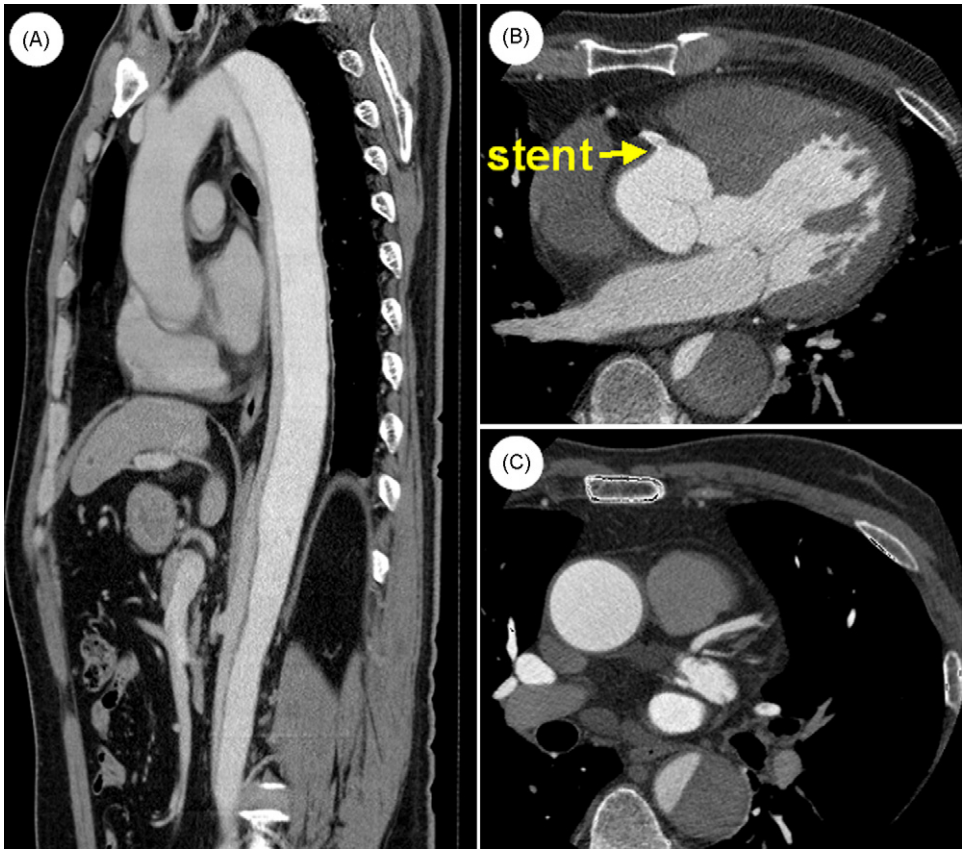

Figure 4 Images obtained by a 64-multislice computed tomography (CT) one year later. (A) An image shown in the sagittal view. (B) The implanted bare metal stent (arrow) was observed clearly at the level of the right coronary cusp. (C) A CT scan showing complete resorption of the ascending aortic intramural hematoma, and showing a descending aortic intramural hematoma.

with AMI. Furthermore, our case demonstrates the need for careful, repeat follow-up imaging, as surgical repair may be required in cases with complications.

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